Date Hb. % (Dare)	Red Cells	White Cells	Neutro philes	Eosin.	Baso-philes	Lymph.	L. Monos. & Transit.	Abnormal Forms
First Entry:								Aniso. Poik.
Aug. 15 38	5,272,000	27,700	54.8	.4	.4	34	10.4	Pallor
Aug. 26 32	5,208,000	19,300	56.5	1.	1.	37.5	4.	Aniso. Poik.
Second Entry:								Aniso. Poik.
Oct. 11 35	4,448,000	19,450	31	4.5	2.5	54.	8.0	.7 Myelo-
Oct. 18 32	7,832,000	19,550	35	6.5	2.	47.	9.5	cytes
Nov. 4 45	5,760,000	11,800	30	0	1.	49.	19.	3 Myelo-
Nov. 18 50	5,952,000	13,000	29	0	0	42.	25.	cytes
Nov. 21 70	7,040,000	9,300	50	0	1	9.	41	0
Dec. 23 65	6,336,000	15,800	26	.9	0	30.	43	0
Jan. 5 63	5,488,000	12,960	29	1	0	45.	25	0

Discussion: This blood picture is rather unusual on account of the high red cell count and low hemoglobin. The differential count was practically normal except for the occasional presence of myelocytes; the moderate change in the size and shape of the red cells and the marked lack of hemoglobin were most interesting. There was marked anisocytosis and poikilocytosis and occasional stipling.

During the past few years a number of cases in infants and young children with this type of blood which resembles closely the blood of chlorosis, found in young adolescent girls, have been reported. Bunge and Abderhalden have shown that a diet containing too little iron can produce in young animals a condition very similar to that of chlorosis. In French literature one finds frequent descriptions of chlorosis in infants. Nonat (Traite de la Chlorose, Paris, 1864) as early as 1864 reported a series of 68 cases with this type of chlorosis. Halle and Jolly (Arch. de Med. des Enfants, 1903) described the blood picture of chlorosis in infants, and Schwarz and Rosenthal (Arch. Ped., 37:1) have collected a series of 40 cases, 29 of which occurred under the first year. This type of anemia shows nothing characteristic in the appearance of the child beyond a marked anemia and diagnosis of the type is not made unless the blood is carefully studied. It is interesting to note that this type of anemia occurs more often in children who were premature or are delicate. It also occurs in twins and those who have had a rather stormy history during the first three months of life. It may occur in the breast-fed as well as in the bottle-fed. It is not limited to girls as is the chlorosis of adolescence, as more cases have been reported as occurring in boys than girls during infancy. The family history often brings out the fact that chlorosis has existed in the family before or that some other type of anemia has been present in other members of the family.

There is usually a loss of appetite, and intestinal derangement associated with this type of anemia during infancy. The mucous membranes are usually pale but not excessively white. Some of the authors speak of a greenish color though this even in the chlorosis of adolescence is not common, at least in the chlorosis that is seen nowadays. The blood examination shows a color index between 0.4 and 0.6; the red blood cells vary between 4,000,000 and 6,000,000; white blood cells may be normal in number, slightly reduced or slightly increased. The platelet count is normal ranging between 200,000 and 300,000. The blood volume is usually normal. Schwarz and Rosenthal estimated that there was a negative balance of iron but a positive balance of nitrogen. Just how much the iron balance has to do with hemoglobin is not known. It is supposed that the premature and weakling has an insufficient amount of iron deposited during foetal life as a greater proportion of the deposit of iron occurs during the last three months of pregnancy. These infants bring into the world a diminished quantity of iron. In older cases intestinal conditions or dietary errors may be a factor in the production of this type of anemia though it would appear that some other factor in addition to diet must be found to account for these cases.

Treatment: This child responded very rapidly to injections of iron citrate. He received bi-weekly injections of 1 c. c. of the citrate of iron. The dietary treatment is of equal importance as that of the administration of iron. Diet should contain liberal amounts of meat juices for young infants and scraped meat for older infants. If freshly-cooked liver can be given, this adds a very definite stimulus to the red blood-forming tissues, combined with spinach, which also stimulates the formation of red blood tissue, which combination has been found most advantageous in the treatment of these secondary anemias. It usually takes from 3 weeks to 3 months before a normal blood picture is re-established, though as has been pointed out in the other cases of anemia reported, the length of time which it takes to recover from a secondary anemia depends somewhat on how long the anemia has continued. The longer a secondary anemia has persisted the slower usually will be the recovery to normal and in some cases the level of regeneration of the blood-forming organs may be very slow in returning to a normal level. In these cases a subnormal level has apparently been struck, over which it is very difficult to bring the blood.

ACUTE PERFORATION OF DUODENAL ULCER*
With Report of Eight Cases

By FRANK J. GUNDRY, M. D., Bakersfield.

This paper comprises a report of acute perforations of duodenal ulcer operated on by me in the past nine years at the Mercy Hospital, Bakersfield.

Acute perforation of duodenal or gastric ulcer is the most serious upper abdominal catastrophe we are called on to treat. The onset is always sudden, the course rapid, and unless a timely operation is done there is a fatal ending in about 90 per cent., according to Deaver. This is probably somewhat high as we often see cases coming to operation later in which the perforation has been closed off by massive adhesions. A careful anamnesis will nearly always bring out the fact that the patient has had previous gastric symptoms, pain coming on three or four hours after eating, relieved by eating, vomiting or alkalis.

The first symptom of perforation is sudden acute, agonizing unendurable pain. Patient lies absolutely still, refusing to be moved in any position. There may or may not be vomiting. Almost immediately after the perforation the abdominal muscles become intensely rigid and, as Deaver states, there is no condition in the upper abdomen where rigidity is so early and marked as in perforated ulcer. This rigidity usually is most marked in the right upper quadrant where also

* Read before the Kern Co. Med. Society March 18, 1921

pressure elicits the greatest tenderness. After perforation, in a few hours, as a rule the symptoms are those of a general peritonitis, when the diagnosis is then more difficult to make.

Whether it is possible to make a definite pre-operative diagnosis or not it is always apparent that a serious intra-abdominal calamity has occurred and the indications are for an immediate operation.

I would call your attention to the early diagnosis and immediate operation in these cases as the mortality bears a definite relation to the time that elapses between perforation and operation. With the exception of two cases, I operated in from two to six hours following perforation.

In all these cases I closed the perforation and did a primary, posterior, gastroenterostomy, but I feel that in my second case the outcome might have been different had I done only a closure of the perforation. In the future in all late perforations, and condition of patient fair, I would limit myself to only a closure of perforation with drainage.

Only in my first and fifth cases had I seen them previous to operation. In these two cases I had seen them several times previous to perforation. Had diagnosed duodenal ulcer and advised operation, which was refused until perforation. The other cases had been treated from three months to ten years for chronic stomach trouble, no exact diagnosis made or operative procedure advised. I think we should learn from these cases that we have been overlooking the pre-perforative diagnosis of duodenal ulcer.

Case 1. J. B., age 28, male, cook.

History of chronic stomach trouble. Present attack commenced with a sudden, excruciating pain in the epigastric region. Abdomen very rigid, especially in right upper quadrant. Operation showed a perforating ulcer of the duodemum. Perforation sutured. Posterior gastroenterostomy. Convalescence delayed by an attack of lobar pneumonia.

Case 2. Q. H., male, age 46.

Stomach trouble for ten years. Two days before admission to hospital was taken with severe pain in pit of stomach. Examination: Abdomen tense, boardlike. Operation showed perforated duodenal ulcer. Closure of perforation, gastroenterostomy. Patient died following day of general peritonitis.

Case 3. J. C., age 36, oilworker.

Chronic stomach trouble, typical of duodenal ulcer. Was taken with severe pain in upper abdomen three hours before admission to hospital. Abdomen boardlike, especially above. Immediate operation. Perforated duodenal ulcer. Perforation closed. Posterior gastroenterostomy. Uneventful recovery.

Case 4. A. A., age 23, farmer.

History typical of duodenal ulcer. At 12 o'clock at night taken with agonizing unendurable pain in upper abdomen. Abdomen rigid, especially over right rectus, high up. Operation three hours later. Perforated duodenal ulcer. Perforation closed. Posterior gastroenterostomy. Uneventful recovery.

Case 5. G. D., age 25, male, blacksmith.

Chronic stomach trouble, pain coming on four to five hours after eating. Was taken about midnight with severe stabbing pain in epigastrium. On examination boardlike rigidity in upper abdomen. Operated four hours after attack and perforated duodenal ulcer found. Ulcer closed and posterior, gastroenterostomy done. Recovery uneventful.

Case 6. G. C., age 36, male, farmer.

Had had stomach trouble for three months when he was taken with severe pain in upper abdomen, every movement causing pain. Entire abdomen rigid, most marked over right hypochondrium. Operated two hours afterwards. Perforating duodenal ulcer. Perforation closed. Posterior gastroenterostomy. Convalescence retarded by slight infection in abdominal wall.

Case 7. P. H., age 36, male.

Had chronic stomach trouble for six years. At 4 a. m. was taken with severe pain in upper abdomen. Unable to move, due to great pain, vomited. Abdomen rigid and boardlike. Operation six hours later. Perforated duodenal ulcer. Perforation closed, posterior gastroenterostomy. Uneventful recovery.

Case 8. E. R., age 49.

Chronic stomach trouble for ten years. Typical of duodenal ulcer. Was taken with severe stabbing pain in epigastrium, unable to move. Abdomen boardlike rigidity. Operated two days following attack. Small perforated duodenal ulcer. Perforation closed, posterior gastroenterostomy done. Convalescence slow due to a sloughing of fascia over rectus muscle.

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Transferred

Dr. H. J. Willey, from Tulare Co. to Fresno Co.; Dr. H. G. Hummel, from Los Angeles Co. to Imperial Co.; Dr. R. J. Sewall, from San Francisco Co. to Los Angeles Co.; Dr. Edward Brigham, from Kern Co. to Tulare Co.; Dr. Harold P. Hare, from Fresno Co. to Los Angeles Co.; Dr. Nelson W. Janney, from Santa Barbara Co. to Los Angeles Co.; Dr. Clarence E. Ide, from San Bernardino Co. to Los Angeles Co.; Dr. Vard H. Hulen, from San Francisco Co. to Alameda Co.; Dr. A. L. Munger, from Sacramento Co. to San Francisco Co.; Dr. C. B. H. Hanvey, from Sacramento Co. to Alameda Co.; Dr. Roland B. Tupper, from San Francisco Co. to Fresno Co.; Dr. John L. McDaniel, from Los Angeles Co. to Merced Co.; Dr. Carl Weltman, from Los Angeles Co. to Fresno Co.; Dr. N. Fujimori, from Los Angeles Co. to San Francisco Co.

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